Motor Systems

Introduction

This is the first of seven lessons on tracts. We'll start with the somatic motor system because it is easier to comprehend; only one cortical area is involved—one primary motor cortex in each hemisphere—and because the motor cortexes mirror each other, we can describe the whole system by following just one side. Additionally, because we move a lot during the day, the somatic motor system tends to be the most intuitive. Technically, any signal from the cortex down runs along an efferent tract, and efferent is synonymous with motor. In this case, motor means somatic movement. Motor impulses travel from the cortex to the periphery. There are several complicated integrated nuclei in the frontal cortex, complicated computing power in the basal ganglia, and a regular assess-and-alter smoothing of movement via the cerebellum. NONE of that is what we're talking about here. We are establishing the concepts of nuclei and tracts, concepts that you are going to use over and over again in different systems.

To keep it simple, this lesson is about the final output of cortical processing, not how it is processed. We are keeping this discussion focused on somatic movement—voluntary movement through the contraction of skeletal muscle. We covered autonomic motor control in General Pharmacology and throughout the organ systems. We also touched on this system in General Physiology. Back then, we were focused on the neurotransmitters and skeletal muscle. Now we're focused on the central nervous system.

Motor Cortex & Homunculus

The **primary motor cortex** is located in the **frontal lobe**, just anterior to the central sulcus. The **central sulcus** separates the frontal lobe and motor cortex (which are anterior to the sulcus) from the parietal lobe and sensory cortex (which are posterior to the sulcus). The primary motor cortex, the location of motor neuron **cell bodies**, makes up the entirety of the precentral gyrus. It starts medially and inferiorly within the Sylvian fissure, follows the superficial contours of the cortex, then rounds the edge of the hemisphere and terminates at the corpus callosum. Therefore, all the cell bodies of these neurons are on the outside of the cortex, and they project their **very long axons** deep into the brain, through the internal capsule and past the basal ganglia, continuing on until they synapse with their target second-order neuron—a motor neuron—in the spinal cord.

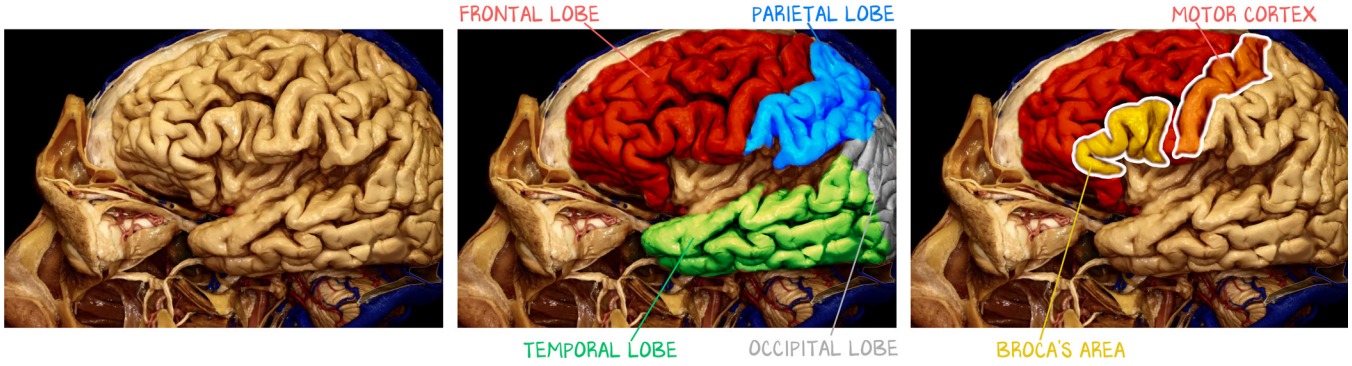

Figure 1.1: Primary Motor Cortex and Frontal Lobe
The first panel is for orientation. We are looking at the lateral aspect of the left hemisphere, with the temporal lobe retracted, the front of the brain on the left, and the occiput on the right. Each panel then identifies specific regions—frontal lobe, parietal lobe, temporal lobe, and occipital lobe—or draws attention to spatial similarities—Broca's area, the region of the brain responsible for moving the muscles of speech, is adjacent to the region of the motor cortex that controls the muscles of eating.

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The motor homunculus represents the different degrees to which muscles are represented in the motor cortex. The larger the area occupied by a structure, the more neurons are dedicated to it. **More neurons mean finer motor control.** Specifically, the greatest numbers of neurons are committed to the face (primarily the tongue, lips, and jaw) and hands—the muscles that perform the most difficult and coordinated movements. Neurosurgeons mapped these points while patients were undergoing neurosurgery. Point stimulation in the hand and speech motor areas can, on rare occasions, cause the contraction of a single muscle. Most often, stimulation causes the contraction of a group of muscles. In other words, the excitation of a single motor cortex neuron usually excites a specific movement rather than one specific muscle. To do this, it excites a "pattern" of separate muscles, each of which contributes its own direction and strength of muscle movement. This should not come as a surprise, as we already covered the concept of a motor unit in General Physiology. But even from the start of the motor signal, that pattern is in place.

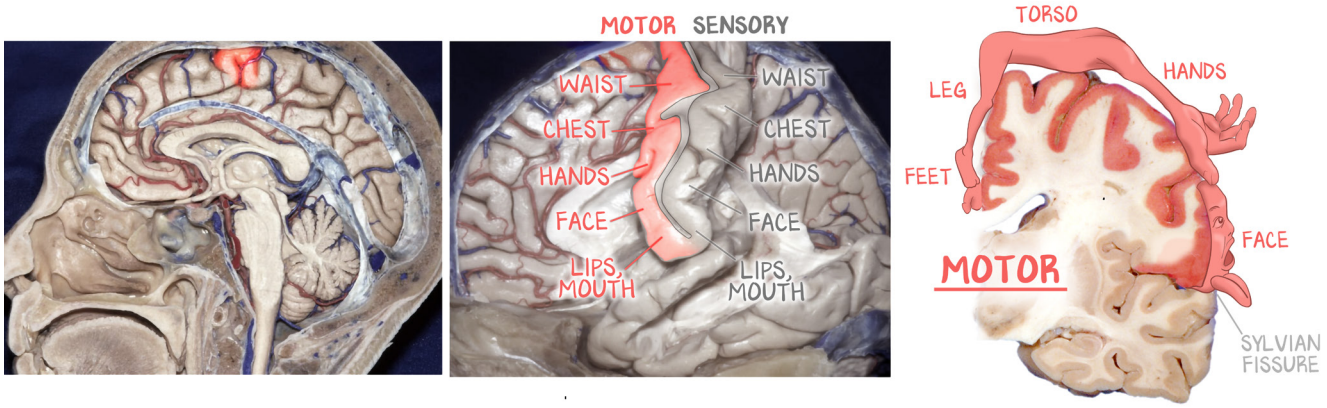

Figure 1.2: Primary Motor Cortex: Homunculus
The primary motor cortex has a topographic representation of the body. The more superior the muscle in the body, the more lateral its representation in the primary motor cortex (more or less). The more inferior the muscle in the body, the more medial its representation in the primary motor cortex. The more surface area dedicated to a muscle group, the larger its representation in the homunculus—the tongue, face, and hands have the most neurons dedicated to them. The illustration on the left demonstrates a mid-sagittal view, showing that the primary motor cortex is the medial portion of the precentral gyrus, does not involve the cingulate gyrus, and wraps around to the lateral surface. The second illustration shows the same mid-sagittal view, but with the primary motor cortex (in red), primary somatosensory cortex (not highlighted), and temporal lobe left in place to maintain the natural anatomy.

This concept of motor patterns has implications for speech. **Broca's area** is anterior to the primary motor cortex, and is also immediately adjacent to the homunculus areas in the primary motor cortex that control the tongue, lips, and jaw. Broca's area is responsible for the formation of the sounds of speech. If a stroke lesioned Broca's area, the patient would be able to hear, understand, and formulate the thought of speaking, but then be unable to actually speak.

We will cover the cerebral vasculature in a future lesson. But understand that because the primary motor cortex is the precentral gyrus, it is perfused by the **anterior cerebral artery**, and thus the ACA is responsible for the feet and legs. Because the motor cortex is the most lateral precentral gyrus, it is also perfused by the **middle cerebral artery**, so the MCA is responsible for the face, hands, and Broca's area. Because the motor cortex shares two vascular supplies, there is also the opportunity for **watershed infarcts**. And the homunculus has another implication for stroke. This is the most nebulous to define, but there must be a transition from ACA to MCA, somewhere between the areas that control the knee and shoulder—the watershed area.

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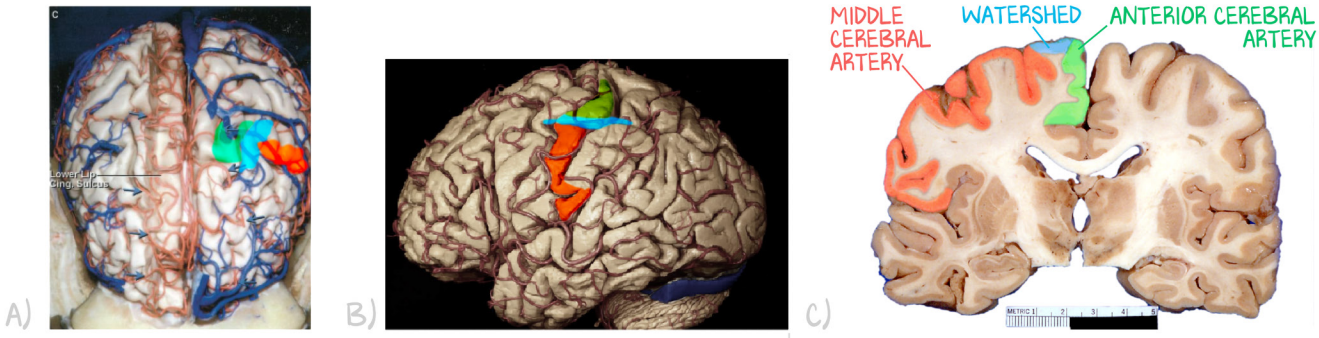

Figure 1.3: Blood Supplies of the Frontal Lobe and Motor Cortex
(a) Superior view of the brain. On the left side of the image, the medial frontal lobe has been removed to show the arteries penetrating the sulci. The right side of the image (left side of the patient) shows the relative contributions of the anterior cerebral artery and middle cerebral artery from this perspective. (b) This panel demonstrates how the middle cerebral artery is responsible for the majority of the lateral motor cortex, as well as the watershed from a lateral view. (c) This panel demonstrates the anterior and middle cerebral arteries' territory of the motor cortex (representational, not exactly anatomic) and the watershed area between them.

Premotor cortex nerve signals cause much more complex "patterns" of movement than the discrete patterns generated in the primary motor cortex. For instance, the pattern may direct the position of the shoulders and arms so that the hands are properly oriented to perform specific tasks. To achieve these results, the most anterior part of the premotor area first develops a "motor image" of the total muscle movement that is to be performed. Then, in the posterior premotor cortex, this image excites each successive pattern of muscle activity required to achieve the movement. This posterior part of the premotor cortex sends its signals either directly to the primary motor cortex to excite specific muscles or, often, by way of the basal ganglia and thalamus back to the primary motor cortex.

While there are many more centers involved with movement, the ones that are worth talking about for now, for comprehension of motor tracts, are Broca's area, premotor cortex, and primary motor cortex. We now leave the cerebrum and talk about the motor tract itself.

The Path through the Brain and Brainstem

Motor neuron axons travel on the ipsilateral side through the brainstem, then decussate (switch sides) within the pyramids in the medulla and continue down the spinal cord on the contralateral side. The **motor cortex controls the contralateral body**. We're going for a deep dive, so ensure to use the figure along with the text.

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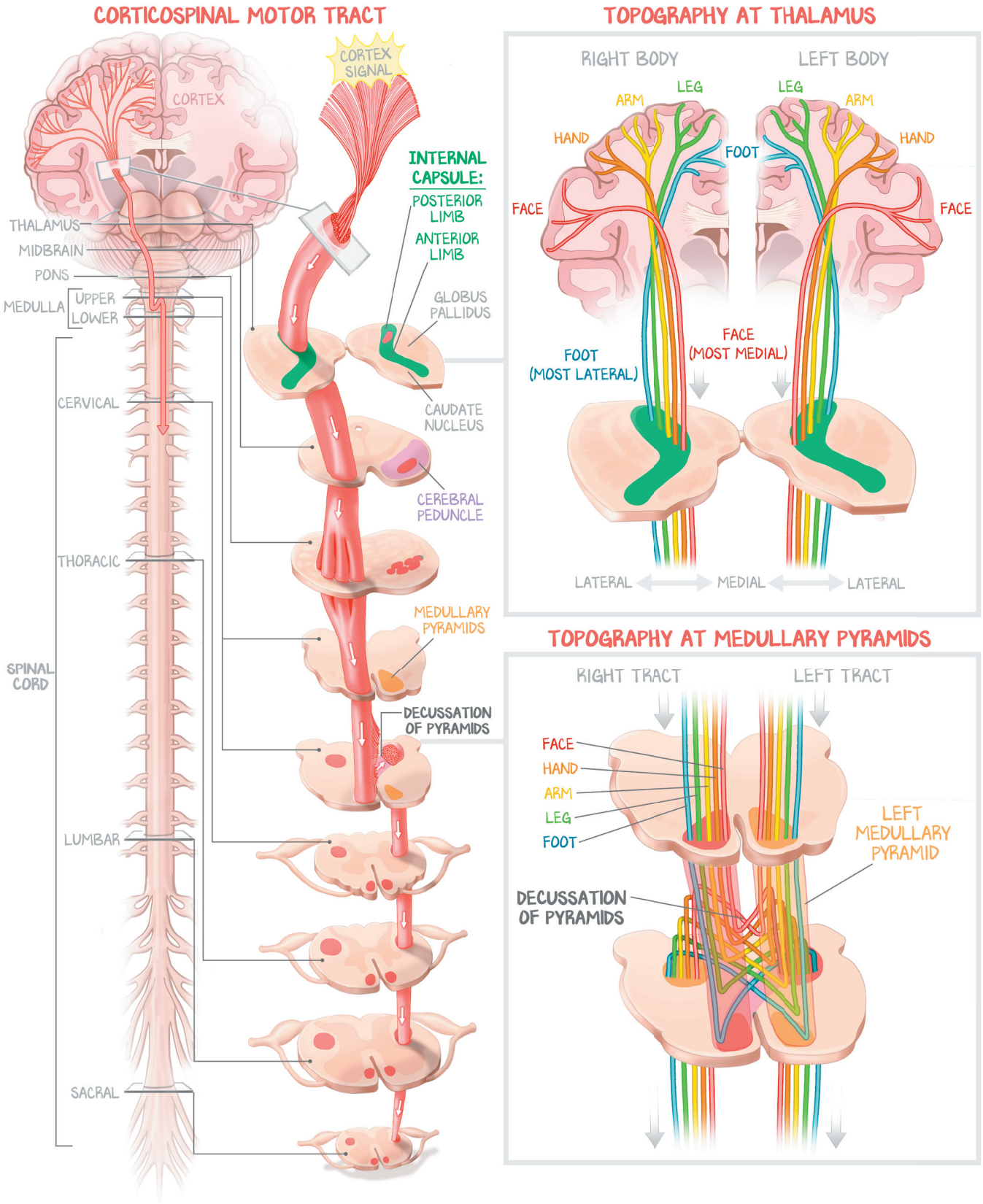

Figure 1.4: Corticospinal Motor Tracts

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A signal starts in the cortex and descends the spinal cord. The motor tract is a **descending** spinal tract. This will have implications when assessing the spinal cord at various levels. You saw **upper motor neurons** in Gen Phys. The neurons in the **grey matter**, on the outside of the motor cortex, are those upper motor neurons. They project their axons into **white matter**, where those axons are cared for by oligodendrocytes, astrocytes, and microglia. Essentially, all of the descending motor fibers (and all ascending sensory fibers) pass through the **internal capsule**. The caudate nucleus (part of the striatum of the basal ganglia) sits medial to the internal capsule, against the ventricles. The motor fibers run through the **posterior limb** of the internal capsule, between the thalamus and putamen/globus pallidus. There is a topographic representation of these motor neurons. They enter the posterior limb of the internal capsule with the **head most medial** and continue that top-down approach so that the **feet are the most lateral**. They will maintain this topographic arrangement through the crura cerebri—medial is the head and face, lateral is the feet and toes. It retains this orientation on each side, as it traverses the pons.

The **corticospinal tracts ARE the medullary pyramids**. The bumps on the anterior of the medulla are not triangular in shape. They are more cylindrical than pyramidal, a round shape being the most efficient. The "pyramids" are within the pons, but they are covered up by all the cerebellar tracts of axons. The "pyramids" are visible under the pons, within the medulla. The "pyramids" taper, becoming progressively slimmer until the only structure that remains is the spinal cord. As axons synapse on their second-order motor neuron, their axonal projections end, and below that, fewer axons continue. Also, not every signal in the motor tract goes to the spinal cord—many go to coordination centers in the brainstem, or they are on their way to move the muscles of the face—all synapses above the brainstem. Thus, as a descending tract descends, it gets progressively smaller.

In the **medulla**, the corticospinal tracts **change sides**. The axons of the motor neurons started on one side. Now, at the distal medulla, they cross over within the pyramids and enter the other side of the spinal cord as the **lateral corticospinal tract** (we're going to call this the lateral corticospinal tract in this lesson because it helps explain the topography, but will call it only the corticospinal tract in subsequent lessons, as we won't talk about any other). The top-down topography is sustained—the **head is first to cross**, and enters the unoccupied medial position left by its partner, the contralateral axon, which crosses as well and takes its place at the most medial. Therefore, in the lateral corticospinal tract, the head is still the most medial. From medial to lateral, the axons are from the nuclei represented by the homunculus, head to toes. Thus, being in a lateral tract with the most medial needing to exit first, the arrangement allows for the motor neurons innervating more superior structures to leave the tract first.

Motor Neurons within the Spinal Cord

White matter is myelinated axons, and grey matter unmyelinated neurons. The basal plate in the developing fetus is the anterior of the spinal cord. The basal plate develops into the motor neurons. This all tracks from the embryology lesson. The upper motor neuron axons will travel within the **lateral corticospinal tract** until they reach their vertebral level. Those axons that leave the lateral corticospinal tract synapse on **lower motor neurons** at that vertebral level. That is the end of that motor neuron's axon. It does not go anywhere else.

The significance of this is that it can be used to figure out which level of the spinal cord you are at just by looking at a sliced section. The farther down the spinal column you go, the more motor neurons will have left the lateral corticospinal tract, the fewer axons there will be, and the less myelin. That means the **proximal lateral corticospinal tracts are large,** whereas the **distal lateral corticospinal tracts are small**. It is the same for sensory fibers but in reverse: the first spinal cord level (the most distal) receives the first axons, so has the fewest axons and accumulates more axons the higher in the spinal column the tract goes. In both cases, the most white matter is seen in the cervical region and the least in the sacral region.

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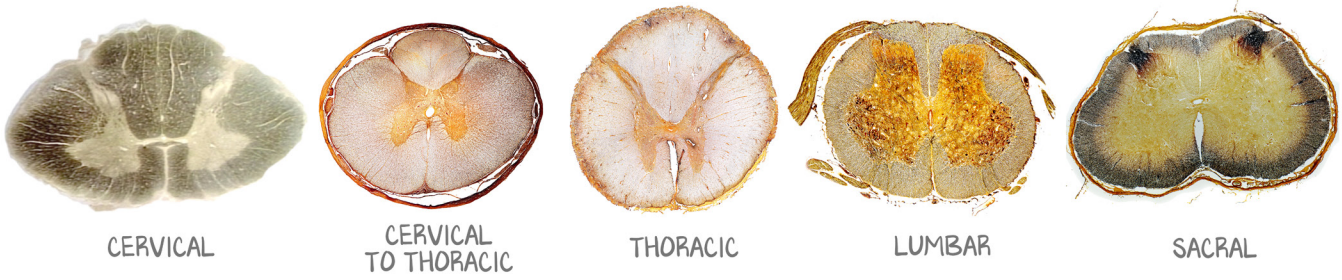

Figure 1.5: Spinal Cord at Various Vertebral Levels
Different stains at various vertebral levels demonstrate less white matter as you move left to right, down the spinal cord. The relative size of the cord also decreases, but we have adjusted these sections so that the smaller cord segments (sacral) are enlarged to fit the height of the illustration.

Within the **anterior horn** of the spinal cord, the anterior grey matter, are the nuclei of lower motor neurons. The lower motor neuron cell bodies are within the spinal cord, so they are derived from neuroectoderm. They have myelinated projections that leave the anterior horn and enter the **peripheral nerve**. The outgoing motor fibers mix with the incoming sensory fibers of the dorsal root ganglion in the peripheral nerve. That motor neuron innervates its motor unit (and back to Gen Phys lessons we go).

Did you notice all the OTHER white matter in the anterior half of the vertebrae? That is all motor tracts from somewhere other than the cortex. These have been traditionally called "extra-pyramidal," as they are everything motor that does not pass through the medullary pyramids. Movement requires a lot more than just the prefrontal cortex planning it and the primary motor cortex carrying it out. It turns out that motor isn't anterior, and sensory isn't posterior. **Cortical motor** is anterior and **cortical sensory** is posterior. The spine sends sensory signals to the thalamus, cerebellum, tectum, and on and on. But for clinical medicine, we're going to stick with saying that there are three tracts, not 27—one motor (lateral corticospinal) and two sensory (DCMLS and STT—the next lesson).

Briefly, in review, the first-order neuron synapses on the second-order neuron. The first-order neuron is excitatory and induces the second-order neuron to depolarize. The second-order neuron innervates its motor unit. The action potential is carried on the typical myelinated peripheral nerve axon. Depolarization at the motor endplate releases far more neurotransmitter than is required to activate nicotinic acetylcholine receptors, resulting in a conformational change and equal conductance to sodium and potassium. Because the density of these channels is high, depolarization of the skeletal muscle occurs, followed by contraction.

Orientation of the Cortex

The cells of the motor cortex are organized in vertical columns that are a fraction of a millimeter in diameter, with thousands of neurons in each column. Each column of cells functions as a unit, usually stimulating the neurons in the anterior horn that innervate a group of synergistic muscles, but sometimes stimulating just a single muscle.

The neurons of each column operate as an integrative processing system, using information from multiple input sources to determine the output response from the column. Following with the trend that many inputs to one output requires many dendrites and just one axon, the **cortex motor neurons are pyramidal**. That is how the medullary pyramids got their name. The reason medical science doesn't equate the corticospinal tract with the medullary columns is that there are many cortico- (from the cortex) and bulbo- (from the brainstem) spinal tracts. We want you to learn the one corticospinal tract. Each column can function as an amplifying system to stimulate large numbers of pyramidal fibers to the same muscle or synergistic muscles simultaneously. This ability is important because the stimulation of a single pyramidal cell can seldom excite a muscle.

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The Reflex Arc

When a lower motor neuron innervates muscle, it is activated by a depolarizing stimulus from its associated upper motor neuron. But that isn't the only way to influence that lower motor neuron. Reflexes are **involuntary muscle movements** induced by a stimulus. Usually, that is a sensory axon of the same vertebral level synapsing onto the motor neuron or an intervening interneuron. And remember, these cells—both motor and sensory—are unipolar. That means some sensory axons leave the dorsal root ganglion and never ascend to the cortex. And the motor neuron only leaves at its vertebral level. So if there were ever a transection of the spinal cord, the reflex arc would still be intact, even if the perception of sensation and voluntary motor control were lost.

The simplest reflex arcs are the **deep tendon reflexes**. The one people are most familiar with is the patellar tendon reflex, the knee-jerk reflex. An examiner strikes the patellar tendon with a hammer (or their fingers). The tendon is stretched, and that stretch is sensed by the **muscle spindle**, which initiates action potentials in **sensory axons** that propagate towards the dorsal root ganglion. It passes along the bifurcated axon of the sensory neuron and into the spinal cord through the posterior horn. The action potentials of the stimulated neurons terminate at **motor neurons** on the ipsilateral side. The motor neurons, in turn, depolarize and activate a muscle contraction. The brain is unaware of the tendon strike and the initiation of contraction because the reflex arc—dorsal root ganglion to motor neuron—is so much shorter than the pathway for the perception of sensation—dorsal root ganglion to cortex. Take a look at the left panel of Figure 1.6, then continue to read.

It isn't quite that simple, however, as there are also **interneurons**. Interneurons are also the target of sensory neurons of the same spinal level. Interneurons of the patellar tendon reflex initiate an inhibitory discharge on the ipsilateral antagonist muscles. The quadriceps need to contract, so the hamstrings are inhibited. There are still even more complex reflexes that involve crossing the midline, but we're not going to cover those. And still, there are more to and from the cerebellum—we'll talk about those later.

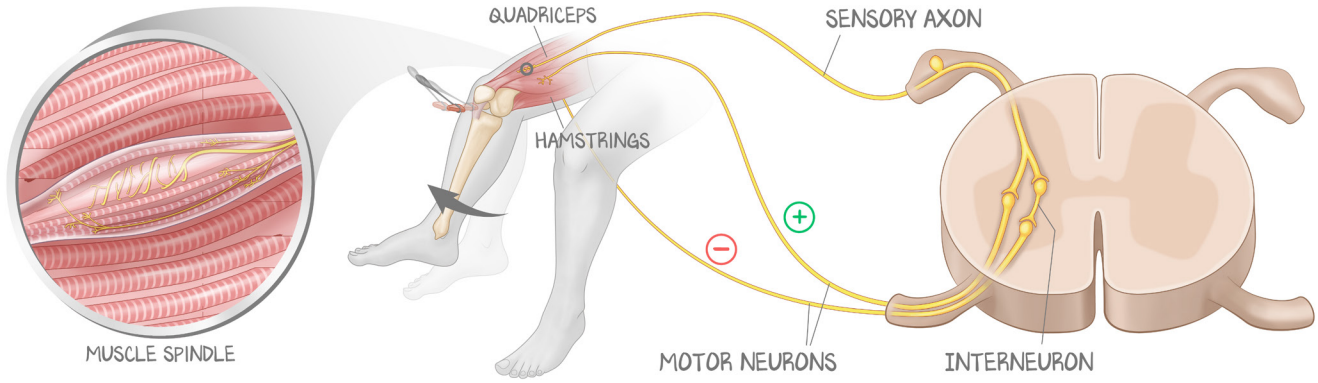

Figure 1.6: Spinal Reflex Arc
As the hammer strikes the tendon, the stretch is sensed by the muscle spindle, which sends a signal to the spinal cord where it activates lower motor neurons to both stimulate the quadriceps and inhibit the hamstrings.

Lesions of Motor Nerves

We discussed this briefly in General Physiology and said we'd spend more time on it in Neuroscience. Well, here we are. There are upper motor neurons and lower motor neurons.

The upper motor neuron has its body on the external surface of the cortex and has a long axon that traverses the brain, brainstem, and spinal cord. The entire length is myelinated by oligodendrocytes

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and supported by astrocytes. If there is a lesion at any point along the tract, from the cell body to the synapse, the lower motor neuron will not receive the signal. The patient will experience an **upper motor neuron lesion**. A "lesion" may be temporary (e.g., demyelination) or permanent (trauma, transection, stroke). It doesn't matter what was lost or where, whether it was the motor neurons themselves in their nuclei in the cortex or it was the axonal tract along the way. From the perspective of the lower motor neuron, it doesn't matter what the etiology is or where the lesion occurred. The lower motor neuron knows only that it doesn't receive a stimulatory signal from the upper motor neuron of the corticospinal tract. From the perspective of the patient, the etiology and location will have other signs and symptoms. Regardless of what those symptoms are, they will include the inability to consciously move the muscle that lower motor neuron innervates.

From your perspective, it does matter where the lesion occurred. You will be tasked with identifying the signs and symptoms of neuron loss and diagnosing the location. Knowing it is an upper motor neuron lesion is only the start because you won't know right away if the upper motor neuron lesion occurred from the ipsilateral side or the contralateral side. Because neurons cross (decussate) in the medulla, any upper motor neuron lesion found in isolation could be before or after the decussation. You will have to pair signs and symptoms of motor dysfunction with sensory dysfunction and assess motor and sensation at multiple levels (face, arms, legs, for starters).

The symptoms of an upper motor neuron lesion are those of **"hyperactive weakness."** The patient cannot will the movement of their limb (**weakness**), but there is also the loss of motor inhibition to the lower motor neuron. This leads to **increased muscle tone** and **hyperreflexia** (hyperactive). There will also be **upward-flaring toes** on the Babinski reflex test. Newborns have underdeveloped upper motor neurons (they aren't fully myelinated at birth). When you stroke the sole of a newborn's foot, their toes will flare up. In an adult, if there is a loss of an upper motor neuron or the axonal tract proximal to the synapse on a motor neuron in the spinal cord, this reflex behaves as it did at birth—the toes will flare up. Normally, stroking the sole of the foot of an adult causes the toes to curl down.

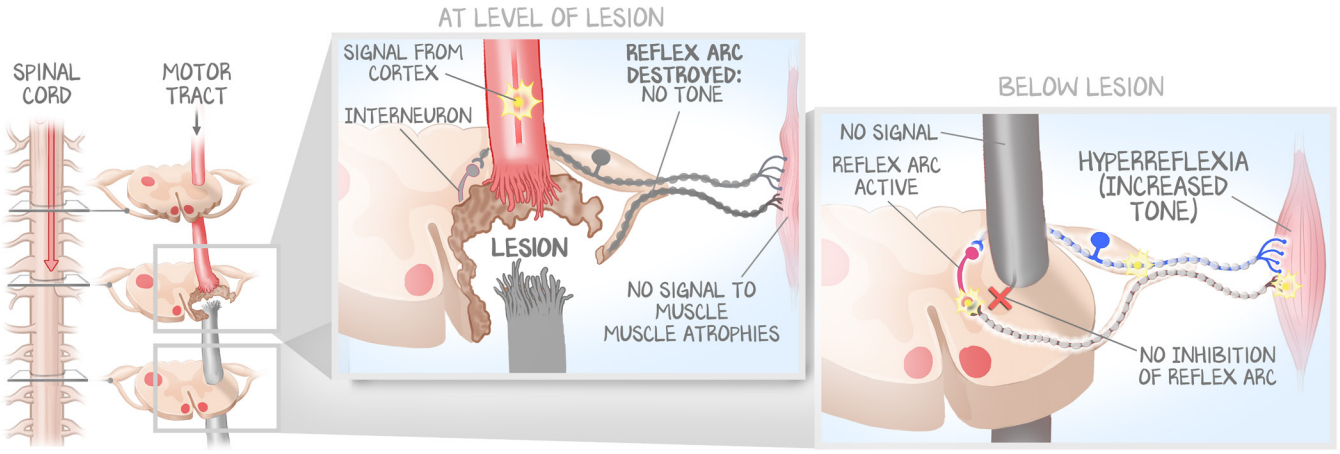

Figure 1.7: Upper Motor Neuron Lesions
When a lesion claims the corticospinal tract, all the motor neurons below the lesion will never receive an action potential from the cortex (or from anywhere else that relies on that region of tissue). There can be no voluntary contraction of the muscle, and no inhibitory signals either. Because the motor neurons below the lesion are alive and well but disinhibited by the lesion above it, the reflex arc is stronger than it was before the lesion. Thus, upper motor neuron symptoms involve weakness (inability to move the muscle) and hyperreflexia (reflex arc intact and disinhibited).

The lower motor neuron has its cell body within the anterior horn of the spinal cord and a long axon that travels within a peripheral nerve. It synapses on the motor unit of the muscles it innervates. The entire length is myelinated by Schwann cells and supported by satellite cells. If there is a lesion at

any point along the tract, from the cell body to the synapse, the *muscle* will not receive the signal. The patient will experience a **lower motor neuron lesion**. A "lesion" may be temporary (e.g., demyelination) or permanent (trauma, transection, stroke of the cord). From the perspective of the muscle, it doesn't matter what the etiology is or where the lesion occurred. The muscle knows only that it doesn't receive a stimulatory signal from the lower motor neuron. From the perspective of the patient, the etiology and location will have other signs and symptoms. Regardless of what those symptoms are, they will include the inability to consciously move the muscle that lower motor neuron innervates. See how similar that was? Just slide it down one vertebral level from lower motor neuron loss, and the rest of the spinal cord down will suffer an upper motor neuron lesion.

As a practitioner of medicine, you will care where the lesion occurred—in the spine or the periphery. For most Neuroscience-related tasks, you will be deducing the vertebral level at which there is a lower motor neuron lesion. Very often, because the lateral corticospinal tracts and the anterior horn are close together, if there is a lesion of the lower motor neuron, there is usually a coinciding lesion of the lateral corticospinal tracts, which means there will be a lower motor neuron lesion at a vertebral level, and upper motor neuron lesions below it. And being a lower motor neuron lesion, it must occur in the spine.

The symptoms of a lower motor neuron lesion are those of **"hypoactive weakness."** The patient cannot will the movement of their limb (**weakness**), but there is also the loss of all lower motor function at that vertebral level. There will be **decreased muscle tone** and **hyporeflexia** (hyporeactive). The muscle will eventually **atrophy**, and there may be the presence of **fasciculations**. Be really careful about the Babinski reflex test with this lesion. If the lesion claimed the lateral corticospinal tracts as well, and the lesion was above the lumbar spine, then there may be **upward-flaring toes** because a lesion at the motor neuron may also claim the tract that neuron belongs to. Anything below the affected lower motor neuron will behave as an upper motor neuron lesion. If the lesion is of the vertebral level that controls toe movement, there may be **no movement at all**. Very rarely will there be the normal downward-curling toes, as that implies a small lesion of the anterior horn that spares the lateral corticospinal tract. It's possible, but unlikely and not part of any of the named syndromes we're going to teach you.

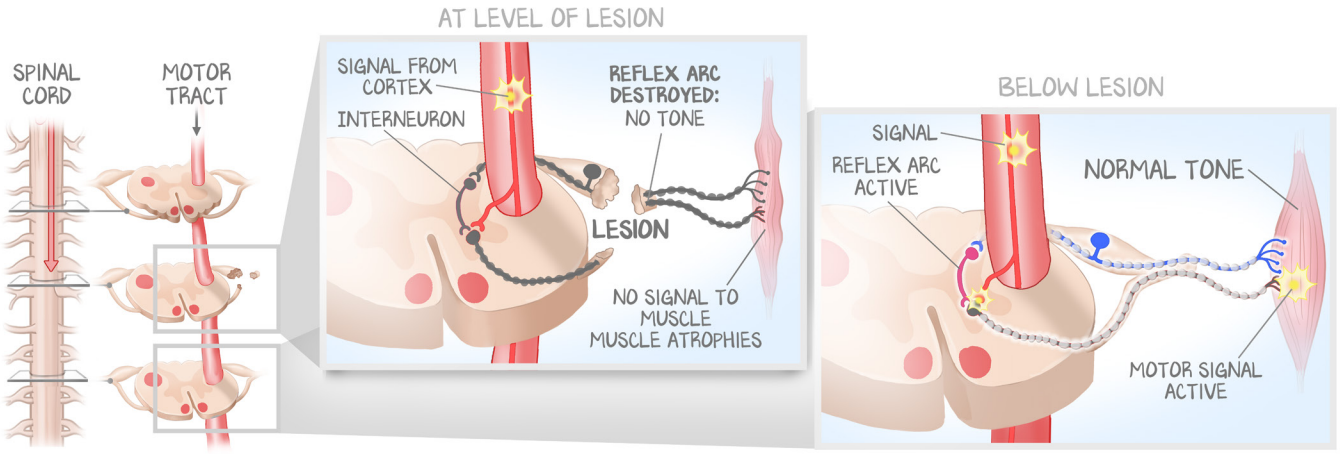

Figure 1.8: Lower Motor Neuron Lesions
A lesion to the peripheral nerve is depicted as a "lower motor neuron lesion" rather than one of the spinal cord as it eliminates the possibility of tract involvement. Conceptually, a spinal cord lesion of the anterior horn has the same motor symptoms as a lesion of the peripheral nerve—no motor action potentials of any kind will reach the muscle, leading to atrophy of the muscle and decreased reflexes. Symptoms below the lesion may be ambiguous—if only the anterior horn is affected and the tract spared, below the lesion will be normal (as depicted); if the anterior horn and the tract are both affected, below the lesion will be as in Figure 1.7.

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Assessing for Level: Motor

Reflexes and strength are the main tools used to assess motor function. You ask the patient to perform muscle movements against resistance (you provide the resistance). **Weakness** (asymmetric strength) alerts you to a problem, and reflexes tell you whether that problem is an upper or lower motor lesion. You will combine this information with what you learned in the sensation lesson to identify the source of the lesion. Above the neck, you test cranial nerves (see the lesson on cranial nerves). Below the neck, you can assess for the **triceps reflex** (C6, C7, radial nerve), **biceps reflex** (C5, C6, musculocutaneous nerve), **brachioradialis reflex** (C5, C6, radial nerve), **quadriceps reflex** (L2–L4), **Achilles reflex** (L5–S2), and the **Babinski reflex** (no localization).

This isn't a lesson on how to perform the maneuvers, but on how to use them. If there are hyperreflexia and weakness, it is an upper motor neuron lesion, and you need to keep ascending until you don't find weakness and hyperreflexia. That enables you to identify the vertebrae between which the lesion has occurred. If you get up into the head and neck, it gets a little harder to keep it succinct, and you should learn the eight presentations in the lesson *Lesions of the Brainstem*.

Other Baby Reflexes

Babies have reflexes when they are born that abate within the first year of life as their frontal lobe develops, and their spinal cord becomes fully myelinated. These are called **primitive reflexes**. Some are useful for assessing weakness (such as the Babinski maneuver). Most are not useful for assessing motor systems in adults because they are extinguished by the development of the frontal lobe and not the myelination of the spinal cord.

The **Moro reflex** causes a baby's arm to spread and then clutch to the chest if its head goes below its shoulders (falling). This would be a sign of an upper motor neuron lesion in an adult, though the maneuver is usually not possible or not worth the effort and risk. The **asymmetric tonic neck reflex** ("fencing" reflex) appears at 1 month and abates by 4 months. When baby's head is turned to the side, it extends the arm in the direction they are turned towards and flexes the opposite arm. This is thought to be a learning of coordination and may represent an upper motor neuron lesion in an adult.

The **rooting reflex** assists with breastfeeding. If stroked on the cheek or lips by any object, baby will turn towards the object. The **suckling reflex** is also linked with breastfeeding, inducing baby to suckle anything that touches the roof of its mouth. The **snout reflex** is when baby purses its lips when tapped on the skin over the philtrum. These tests serve no purpose in assessing strength, but their presence in an adult indicates **frontal release sign**—loss of the frontal lobes.

Citations

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